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CONTENTS

PAGES

Diseases Other than Tuberculosis
d. *Pneumonoconiosis (concluded)* 21–22
e. *Miscellaneous pulmonary conditions* 22–28
f. *Extrapulmonary conditions* 29–35
Prophylaxis and Prevention 36–38

Silicosis Laws in Great Britain and South Africa.—Gold was "proved" on the Rand in 1885 and mining started there the next year. At first the workings were shallow and the amount of dust not excessive but by 1892 extensive deep level mining was being done with increasing use of drills. In 1899, in 100 mines working there were employed 12,000 whites and 100,000 colored persons. The first warning of the injurious effect of mine dust on health was given by the Government mining engineer of the Transvaal Mines Department in 1902. He stated that, of 1,377 machine men employed before the Boer War, 225 were known to have died between October, 1890 and January, 1902. In December, 1902, Lord Milner appointed the first Transvaal Miners' Phthisis Commission and regulations were made and supplemented from time to time. In 1907 the Mining Regulations Commission was appointed to revise and amend the regulations and in 1911 a new code was issued. Compensation for miners' phthisis was now introduced for the first time, in the Miners' Phthisis Allowances Act to provide temporary relief for necessitous cases. In 1912 an act was passed laying down a financial basis on a modified insurance method, there being established both a Miners' Phthisis Compensation Fund and a Miners' Phthisis Insurance Fund. The former made quarterly "levies" on "scheduled" mines to which Government contributions were added. The latter fund, based on wages, required a 2½ to 5 per cent contribution from the mines and 2½ per cent from the miners, benefits to begin from August 1, 1914. In 1915 a Select Committee reported recommendations which were given effect in 1916, including a central medical bureau and periodical medical examinations. The Miners' Phthisis Act of 1919 repealed all previous acts and abolished workers' contributions for compensation funds, also introducing differential rates of levy. A Miners' Phthisis Board was appointed by the Government and determined the amount of levy, granted benefits, reviewed awards and reported regularly to Parliament. A medical bureau was appointed by the Minister of Mines and paid by the Government and their duties included the conduct, direction or control of all medical examinations under the Act, with special provisions in the case of appeal. In 1925 a consolidating and amending act was passed, providing for further appeal from decision of the Bureau to the Medical Board of Appeal, consisting of three practitioners with special knowledge of respiratory disease. In 1934 a Miners' Phthisis Amendment Act made some important changes, especially in requirements for valid claim for benefits, as follows: (*1*) The claimant must have worked under ground in "scheduled" mines for a period aggregating 5 years. (*2*) A portion of that period must have been subsequent to August 1, 1908. (*3*) Claims for benefits must be submitted within five years from the date of last working underground. Certain specified exceptions were made. In 1906 the British Committee on Compensation for Industrial

Diseases considered the possibility of extending such compensation to certain respiratory diseases, including pulmonary fibrosis and fibroid phthisis. Definite legislation providing compensation for silicosis was not introduced until 1919, but in 1917 a special scheme of compensation was worked out for the refractories industry, the group getting and manipulating gannister and other highly siliceous materials. In 1918 the Workmen's Compensation (Silicosis) Act was passed. It provided for the establishment of a general compensation fund to which all employers in the specified industries were required to subscribe, and out of which compensation and expenses should be paid. Settlement of claims was by joint committees with independent chairmen and all medical examinations were by specially appointed medical officers. In 1919 the Departmental Committee on Compensation for Silicosis dealing with the Refractories Industries Scheme made certain important recommendations, including the establishment of a medical board of full-time officers to make all medical examinations excepting certain initial examinations and to give all medical certificates. Persons found to be suffering from pulmonary tuberculosis were excluded from the industry and tuberculosis was not compensated unless accompanied by silicosis. In August, 1924 the recommendations were adopted but revised in 1925. In 1929 a scheme was drawn up for the sandstone industry, in 1927 for the grinding industries, and in 1928 for various others. In 1931 revisions were made and the asbestos industry added. The medical board was increased to nine. Compensation for death or disability due to silicosis, asbestosis, or either or both with tuberculosis, contracted during employment, is now payable in all industries shown to have increased risk of these. Reported cases have rapidly increased. Research is also proceeding everywhere.—*Silicosis at Home and Abroad, Special Article, Lancet, March 27, 1937, ccxxxii, 773.*—(*A. P.*)

Congenital Cystic Disease of Lung.—Aetiology is still unknown. In general, the cases may be divided into those in which there are single or multiple large cysts containing fluid or air, and those in which there is diffuse degeneration resulting in the honeycomb type of lung. Symptoms are not pathognomonic. Recurring attacks of severe dyspnoea with cyanosis, particularly in infants and children, and in older individuals, a progressive dyspnoea with or without previous respiratory infection, should be suspected. Severity of symptoms depends upon the extent of destruction of parenchymal tissue, and change in intrathoracic pressure. Diagnosis is dependent upon the X-ray findings and the introduction of lipiodol may be necessary. Artificial pneumothorax may be necessary to distinguish a balloon cyst from spontaneous pneumothorax. Fluid-containing cysts must be differentiated from thoracic tumor, pulmonary abscess, dermoid, echinococcus cyst, and empyema. Treatment is unsatisfactory. Some cases improve following bronchoscopic aspiration and lipiodol injection. Some fluid-containing cysts have been successfully removed surgically. For diffuse saccular or honeycomb type of bilateral cystic degeneration, no treatment seems worth while. Secondary infection is the greatest danger to which these patients are subject. Surgical measures to occlude a communicating bronchus are a proper procedure as is also removal of fluid-containing cysts. Infected cysts may benefit from bronchoscopic aspirations and injections of lipiodol.—*Congenital Cystic Disease of the Lung, H. G. Wood, Jour. Thoracic Surg., August, 1937, vi, 634.*—(*L. F. B.*)

Total Pneumonectomy for Congenital Cystic Disease.—A case of infected congenital cystic disease of the lung was treated successfully by total pneumonectomy. For lack of precedent to follow, the case was previously treated by artificial pneumothorax, phrenic surgery and thoracoplasty. The condition was improved by these procedures. Vital capacity following thoracoplasty was 20 per cent of normal, increased to 36 per cent the following six months, was 25 per cent following pneumonectomy and increased to 33 per cent six months following this operation. Progress of the disease may be temporarily checked by collapse measures, but ultimate success can only be obtained by complete removal of the diseased process. Cautery drainage might be of value where the infection is limited to a localized area.—*Total Pneumonectomy for Congenital Cystic Disease of the Lung, J. W. Gale, J. L. Kealey & H. M. Coon, Jour. Thoracic Surg., August, 1937, vi, 626.*—(*L. F. B.*)

Pulmonary Pneumatocele.—The propriety of the use of the term "congenital" in the X-ray diagnosis of cystic disease in the absence of previous respiratory history is questioned. It is felt that recently many pulmonary changes have been classified as congenital pulmonary disease. Weller has stated that he can recall no instance of changes being found in the lungs of newborn infants or very young children which would lead to a diagnosis of congenital cystic disease, though one would expect to encounter such occasionally at least. The roentgen

diagnosis of cystic pulmonary disease should more properly be classified as follows: (*1*) true congenital pulmonary cyst or cysts; (*2*) (a) chronic interstitial pneumonitis with emphysema; (b) chronic bullous emphysema; (*3*) cystic bronchiectasis, and (*4*) pulmonary pneumatocele (localized alveolar or lobular ectasia). True congenital pulmonary cysts are rare; evidence of such lesions have been found in very few fetuses or newborn infants. The authors have seen only one unquestionable case in twelve years. Only two cases have been reported in the literature of coexistent air-filled cystic spaces and closed fluid-containing cysts, and it seems unlikely that all cysts in all cases should have ruptured and become aerated. Chronic interstitial pneumonitis with emphysema may be the result of contractures from interstitial inflammatory changes associated with bronchopneumonia or the progressive fibrosis found in some unresolved lobar pneumonias. Accentuated interlobar septa and interstitial scarring outline the dilated emphysematous areas in the roentgenogram which tend to be lobar or unilateral. Chronic bullous emphysema, due to obstruction by bronchospasm as found in asthma, will present the X-ray appearance of multiple air cysts seldom containing fluid or exudate. This process is more generalized and the septa are less prominent than in the chronic interstitial pneumonitis with emphysema; flattening and depression of the diaphragms may be pathognomonic. The large majority of cases which have been reported resemble cystic bronchiectasis or pulmonary pneumatocele roentgenologically and in having a history of previous respiratory infection or contagious disease of childhood. The greater difficulty in aeration of the left lung, that is, the normal tortuosity of the left airway, the lobular character of aspiration pneumonia and the frequency of lobar or bronchopneumonia in infancy and childhood with their accompanying pathology and residual abnormalities, are presented as more logical explanations for the origin of multiple cystic pulmonary disease than congenital abnormality with or without superimposed infection. Two cases are presented demonstrating the development of cystic bronchiectasis following acute respiratory infection in lungs which previously showed no roentgenographic evidence of cystic areas. The fourth group of patients consists of those with pulmonary pneumatocele. They are characterized by single or a few contiguous emphysematous alveoli which tend to increase in size and may assume massive proportions with compression of the surrounding lung. They probably originate as an acute lobular emphysema following a lobular pneumonia and are due to a persistent check-valve obstruction of the bronchial lumen produced either by nonresolution or subsequent distortion by the dilated air spaces. The symptoms are seldom more than cough and dyspnoea on exertion. A bronchogram or diagnostic pneumothorax will demonstrate their intrapulmonary character. Four cases of this type are presented, all developing subsequent to an acute lobular pneumonia. The authors conclude that it is improper to use the term "congenital" in the roentgen diagnosis of cystic pulmonary disease without film evidence of such a lesion at birth; that the congenital origin of most cystic pulmonary disease is doubtful, and that cystic pulmonary disease may be further subdivided on the basis of aetiology and method of development.—*Pulmonary Pneumatocele (Localized Alveolar or Lobular Ectasia), C. B. Peirce & P. R. Dirkse, Radiology, June, 1937, xxviii, 651.*—(*G. F. M.*)

Lung Cysts.—Up to the present time less than 300 cases have been reported, more than half during the last decade. Their specific aetiology is unknown and they have been associated with or ascribed to a variety of conditions. Their congenital origin is assumed by most informed observers. Noninfected cysts may be asymatomatic, perhaps throughout life. Of the 3 cases reported, one, in a 16 year old girl, was infected and lobectomy recommended, as the symptoms were marked; another, in a woman of 57, showed calcification and had symptoms only during a brief phase; the third, in a girl of 16, was asymptomatic and discovered on examination for a camp. Roentgenographically the absence of the usual hilum stump helps to differentiate lung cyst from pneumothorax and a thin wall is usually demonstrable. Bronchographically also the wall should be seen, while bronchoscopy reveals the presence of bronchial stenosis and secretion. Encysted empyema, abscess, and echinococcus cyst should also be considered. Air-containing cysts, with elastic walls, may be aspirated with ensuing recovery. Infected cysts require radical treatment, usually lobectomy. Under three years of age lung cysts carry a high mortality. Roentgenographic reproductions accompany this paper.—*Lung Cysts, L. Rabinowitz & E. J. Rogers, New England Jour. Med., May 27, 1937, ccxvi, 919.*—(*A. P.*)

Congenital Cysts of Lung.—Congenital cystic disease of the lung is a developmental malformation which may involve any portion of the bronchial tree or interstitial pulmonary tissue. The cysts are solitary or multiple and contain a milky or turbid fluid

at birth. Should a cyst rupture into a bronchus it becomes an air cyst, and if the air is trapped by a valve-like orifice the cyst expands. A review of the literature reveals that of the 374 cases reported with an adequate description of the cyst type, 141 were solitary. Cystic disease of the right lung was present in 157 instances, and of the left in 136 instances. In 78 cases it was bilateral. The opinion which prevails concerning the pathogenesis of this condition is that a small pulmonary bud is pinched off and continues to develop into pulmonary tissue. As there is no bronchial outlet the secretions are retained and distention occurs. The wall of the cyst is composed of high or low columnar epithelium with or without cilia; muscle, elastic tissue and cartilage are sporadically arranged through the deeper layers. The diagnosis of cystic disease is based chiefly on the X-ray findings. A history of symptoms present from birth and without any history of severe respiratory disease is very suggestive. In children the symptoms are essentially recurring attacks of dyspnoea and cyanosis with or without cough. In the case of expansile cysts these symptoms may be very dramatic. Unless the cyst is deflated death may occur rapidly. Cysts communicating with a bronchus are prone to become infected. This results in symptoms and a type of sputum resembling that seen in cases of bronchiectasis. In the absence of infection there is no fever. Haemoptysis is common and may lead to a mistaken diagnosis of tuberculosis. This has occurred especially frequently in cases in which a cavity could be demonstrated by X-ray, and in spite of the fact that the sputum contained no tubercle bacilli. Pains in the chest are inconstant, and may be referred to the epigastrium. A wheezing type of respiration may be present. It is important to realize that these cysts may never give rise to symptoms, or not until adult life. Two hundred and seven of 368 cases were not recognized until the patient was past fifteen years of age. A solitary fluid cyst appears on the X-ray film as an opaque globular shadow which lies within the lung. It is slightly less dense than the shadow made by the heart. The material obtained by aspiration of the cyst resembles amniotic fluid. It is slightly milky or albuminous and moderately viscid. In the diagnosis of lung cyst it is important to exclude tumors such as teratoma, chondroma, and ganglioneuroma. This can usually be done by means of a careful history, and physical examination, plus a period of observation. A cyst usually remains stationary as to size. Its border is less clearly defined. If in doubt puncture will clinch the diagnosis. Echinococcus disease may be differentiated by the complement fixation test, the eosinophile count, and the presence of hooklets in the sputum. Dermoids may be differentiated by the demonstration by X-ray of teeth and bone, or by the presence of hair in the sputum. Hydatid cysts and mediastinal cysts arise from the mediastinum, and rarely produce dyspnoea. Differentiation from inflammatory lesions such as pneumonia, encapsulated empyema, interlobar effusion, and lung abscess is usually easy. Solitary nonexpansile air cysts appear within the lung as globular or spherical air pockets. They are thin walled and although they may be trabeculated no pulmonary markings can be seen traversing them. On deep inspiration they produce an increased illumination which can be demonstrated by roentgenoscopy. Expansile cysts produce an obliteration of the pulmonary markings with air filling the entire hemithorax from apex to base. The diaphragm is depressed or flattened, and the costophrenic angle is obliterated. There is often a haziness at the apex. The hilar shadow is elongated and thinned, and the collapsed lung does not appear as a broad stump as is the case in the presence of a pneumothorax. The cyst wall can usually be detected in its mesial portion. The affected side of the thorax may bulge. Puncture releases air under pressure. By means of an induced artificial pneumothorax beautiful X-ray pictures may be obtained in these cases. Multiple cysts are usually spherical, discrete and irregularly arranged. There is no infiltration of the parenchyma between the cysts. This type rarely produces symptoms and what symptoms are produced are present from birth. Sacculated bronchiectasis from a contractive process can usually be differentiated from congenital cystic disease, because in the former the cavities are smaller, less discrete, and more uniformly arranged about a bronchus. Also bronchiectasis commonly first appears following a severe respiratory disease. The diagnosis may remain undecided, however, until at autopsy the presence or absence of pigment in the walls of the cavities can be established. Diaphragmatic hernia can usually be easily distinguished by the use of a barium meal and by the presence of gurgling sounds within the chest. Iodized poppy seed oil may prove of value in diagnosis. Where treatment is required surgical removal of the involved lobe or lung is the only rational procedure.—*Diagnosis of Congenital Cystic Disease of the Lung, S. G. Schenck, Arch. Int. Med., July, 1937, lx, 1.*—(*C. L. D.*)

Vanishing Lungs.—A far advanced case of bullous emphysema is reported. In 1929

the patient, a man of 28 years of age, complained of general weakness and increasing shortness of breath. A diagnosis of tachycardia and chronic bronchitis was made. A year later he was admitted to the Veteran's Hospital where a diagnosis of bilateral idiopathic pneumothorax was made. During the next few years he was a patient at various institutions and always was a diagnostic problem. When last readmitted to the Veteran's Hospital in October, 1935, he was weak, dyspnoeic and moderately emaciated. The physical findings of significance were: blood pressure of 108/96, rapid rather weak heart sounds, emphysematous type chest with hyperresonance, absence of breath-sounds except in the bases, absence of whispered voice and no amphoric breathing. A diagnostic pneumothorax gave neutral readings on both sides. On reviewing the chest films taken over a period of six years a downward loss of lung markings was seen. A circumscribed mass in the left hilum, which slowly increased in size until death, was first noticed in 1935. The patient was subject to acute attacks of dyspnoea suggestive of cardiac asthma. He expired in April, 1936. A postmortem film showed absence of lung markings down to the fifth rib on the right and the eighth rib on the left. When the chest was opened at autopsy the lungs ballooned out and overfilled the chest cavity, especially on the left; both lungs had a lobulated appearance with many blebs seen. The right upper lobe contained one large bleb with a number of smaller bullae and blebs along its lower margin; no communication could be demonstrated between these. A giant bulla occupying four-fifths of the chest cavity and composed of an empty pleural sac was practically all that remained of the left upper lobe. Atmospheric air was present in all the bullae. Some increase in fibrosis was found in the compressed lower lobe but there was no evidence of tuberculosis. The slowly growing tumor seen on the left was a bronchogenic carcinoma, grade IV, which extended along the left main descending bronchus. The bronchi going to the upper lobes were narrowed and occluded though no apparent cause for this was found. Points helpful in the diagnosis of this case were the absence of cough or sputum, unexplained progressive dyspnoea with periodic attacks of severe dyspnoea, pulmonary hypertension, and signs of pneumothorax without the amphoric breath-sounds frequently heard in pneumothorax. The significant findings in the X-ray films were gradual diminution of the lung markings beginning at the apices, absence of the sharp lung line seen in pneumothorax, fine unanatomical markings due to fibrous septa in the involved areas, progression of the affected area and the absence of fluid frequently seen in chronic pneumothorax. Bullous emphysema must be differentiated from tuberculous cavity and cystic disease of the lung. Lipiodol instillation showing stenosed or occluded bronchi is an important procedure and serial films showing progression are pathognomonic.—*Vanishing Lungs: A Case Report of Bullous Emphysema, R. M. Burke, Radiology, March, 1937, xxviii, 367.*—(*G. F. M.*)

Changes in Lungs following Irradiation.—Of a series of 51 consecutive patients, comprising 47 cases of carcinoma of the breast, 3 of recurrent fibrosarcoma of the back and chest-wall, and one of haemangioendothelioma of the supraclavicular region, who received intensive treatment with roentgen rays, radium, and radon, 12 (all carcinoma of the breast) showed postirradiation changes in the lungs and pleura. Six of these showed mild, but definite changes, consisting of parenchymal fibrosis and thickened pleura on the irradiated side. Four others showed more marked changes consisting of fibrosis, thickened pleura throughout with filling up of the cardiophrenic angle, and displacement of the heart and trachea to the affected side. One of these 4 cases had metastatic nodules in the lower part of the lung field while the fibrosis was observed in the upper part where the irradiation had been heaviest. In another of these 4 cases the irradiation and subsequent fibrosis were bilateral. Case 11 had low grade fever and recurrent pleural effusion for more than two months, while case 12 had an increase in pleural shadow and lung markings after the first series of treatments. These later decreased only to increase and be accompanied by pleural effusion after the next irradiation. All sputum and pleural fluid examinations were negative for tubercle bacilli (guinea-pig inocculation was not done), neither were there signs or symptoms of pulmonary tuberculosis, or the infiltrative type of pulmonary metastasis and interstitial pneumonia in these cases. In general, the cases that received the most intensive radiation therapy showed the most marked changes. None of the cases who showed any reaction received less than 1,000 r of roentgen rays.—*Changes in the Lungs and Pleura following Irradiation of Extrathoracic Tumors, C. K. Hsieh & H. T. Kimm, Amer. Jour. Roentgenol., June, 1937, xxxvii, 802.*—(*E. M. J.*)

Pseudocavitation.—Five cases of localized and encapsulated pneumothorax are presented; the roentgenographic appearance is similar to that of cavitation within the

lung parenchyma. The encapsulated pneumothorax which simulates cavity is always found peripherally or in the region of the interlobar septa. Two of the cases were proved by postmortem examination; the remaining three showed an amazing regression or disappearance of the defect after three months of bed-rest. Neither symptoms, physical signs, nor roentgenograms, serve to differentiate satisfactorily parenchymal cavitation from this type of encapsulated pneumothorax.—*Encapsulated Pneumothorax, C. Schumann, Quart. Bull. Sea View Hosp., April, 1937, ii, 221.*—(*A. A. E.*)

Infarction of Lung.—Pulmonary infarction is an important and frequently fatal complication of severe mitral stenosis. The diagnosis is often difficult, especially when there are present symptoms and signs suggesting pulmonary consolidation of rheumatic origin. The occurrence of blood-streaked sputum should always lead one to suspect that pulmonary infarction has occurred. This condition should also be considered whenever, in the absence of other complications, congestive failure does not respond to general routine treatment. The records of 52 patients dying with mitral stenosis were studied from a series of 2,500 records of autopsies at the Massachuseetts General Hospital. Of the 23 cases with death from congestive failure 61 per cent had pulmonary infarction, while of the 29 cases in which death was not associated with congestive failure only 7 per cent had pulmonary infarction. Twenty-one per cent of 39 cases of hypertension in which death was associated with congestive failure had pulmonary infarction. It is important to diagnose this condition because of its grave prognostic significance. Five cases are reported including one with jaundice. The causes of pulmonary infarction include thrombosis *in situ*. The combination of a sluggish pulmonary circulation and arteriosclerosis is likely to lead to such thrombosis. Embolism from concealed thrombosis of systemic veins and embolism from thrombi in the right side of the heart may cause infarction. Acute infection superimposed upon an old rheumatic lesion may also be a source of pulmonary infarction. One must not expect the onset of this condition to be heralded by acute signs. Intractable pulmonary embarrassment without any sudden episode in the presence of severe disease of the mitral valve and congestive failure, with or without a demonstrable friction rub, should always suggest the possibility that pulmonary infarction has occurred.—*Pulmonary Infarction Complicating Severe Disease of the Mitral Valve, Harry B. Levine & Paul D. White, Arch. Int. Med., July, 1937, lx, 39.*—(*C. L. D.*)

Bronchoscopic Investigation of Haemoptysis of Uncertain Cause.—The significance of haemoptysis as a sign of serious pulmonary disease has long been recognized. However, of late years observers have pointed out that haemoptysis was more frequent in some other conditions, for example, chronic bronchiectasis, than in pulmonary tuberculosis. Würtzen (1936) found that of 869 cases admitted to sanatoria on account of haemoptysis between January, 1929 and August, 1935, only 582 had pulmonary tuberculosis. In other words, one out of every three such cases had been wrongly diagnosed. The actual causative lesions were found in 287 and included pneumonia, 50 cases; new growth, 20; cardiac disease, 60, and epistaxis, 58. The development of X-ray technique for chest examination was the first great advance in positive differential diagnosis and next came the perfection of the technique of bronchography with contrast media. Further assistance was rendered by bronchoscopy, especially in cases where nothing was demonstrable by the other methods. In haemoptysis of unknown origin it is indispensable. Six cases are recorded with recurrent haemoptysis without physical or roentgenographic evidence of pulmonary disease in which the causative lesion was discovered by bronchoscopy. They include tracheal varix, chronic granuloma, malignant and benign neoplasms. Most of the literature on tracheal varix is from the European Continent. The only clinical sign is sudden profuse haemoptysis, accompanied by cough, independent of exertion and tending to recur. It is commonest in people of middle age and and the site of predilection is the anterior tracheal wall just below the vocal cord. The Jacksons have thoroughly discussed chronic granuloma (1931). The masses may be either sessile or pedunculated and vary in size and location. Ulceration is rare and also fixation or rigidity of the bronchial wall. The early signs are haemoptysis and a wheeze heard over the open mouth. Chevalier Jackson (1934) wrote of benign tumors as very common and occurring anywhere in the tracheobronchial tree. Wheezing heard at the open mouth and haemoptysis are the commonest symptoms. Adenoma is apparently the commonest type, fibroma next. Bronchoscopy is the only means of diagnosis during life. Malignant neoplasms also are common, their manifestations various and their symptoms suggesting asthma, tuberculosis, unresolved pneumonia, lung abscess, bronchiectasis, etc. Sixty-two per cent occur in the larger bronchi and parenchymal location is rare. A large number are within easy access of the bronchoscope. Haemoptysis again is a common symptom, occurring in 48 per cent. Profuse haemorrhage is apt to be

a late development. X-ray evidence is not available until there is some bronchial occlusion or parenchymal infiltration or glandular metastasis. Contraindications to bronchoscopy are few and dictated by common sense, for example, recent profuse haemorrhage or a moribund condition. Suspected pulmonary tuberculosis is not a contraindication. There is practically no risk involved and much information to be gained. We should be prepared to "look and see" rather than "wait and see."—*Direct Bronchoscopic Investigation in Haemoptysis without Physical or Radiological Manifestations, J. E. G. McGibbon & E. T. Baker-Bates, Brit. Med. Jour., January 16, 1937, no. 3967, 109.*—(*A. P.*)

Atelectasis.—It is convenient to divide the process of obstructive atelectasis into three stages. (*1*) The early or dry stage results from the absorption of entrapped alveolar air by the capillaries. This produces a shrinkage and partial consolidation of the lung, and the physical signs are those of retraction and deficient aeration. Roentgenograms show deviation of the trachea and mediastinum toward the affected side, diminished aeration and slight rise of the diaphragm. (*2*) The wet stage is produced by flooding the alveoli with liquid; at this time the physical signs are those of pleural effusion except for the retraction of the heart and trachea. The roentgenograms show a dense, homogeneous shadow in the affected hemithorax. (*3*) The stage of fibrosis is the end result. The collapse produced by pneumothorax without bronchial obstruction results in a fairly mobile and at least partially aerated lung in which liquid does not accumulate as it does in the lung distal to a point of bronchial obstruction.—*Obstructive Atelectasis, Radiographic Studies of the Dry and Wet Stages, J. Kaunitz, Quart. Bull. Sea View Hosp., April, 1937, ii, 290.*—(*A. A. E.*)

Follow-Up of Pulmonary Fibrosis in Children.—This series comprises 45 children with pulmonary fibrosis observed at two Broadstairs convalescent homes between June, 1928 and August, 1934. Excluding very mild or recent cases there were 29 children circularized through the assistance of the Invalid Children's Aid Association, of whom 16 were reëxamined at a special clinic and reports were received from 7 others. Nearly all the more severe cases were included. The material was quite varied, all manner of physical signs being encountered, and a few even simulated pulmonary tuberculosis, although irregular and basal distribution was more common. With one exception all were in good health and the local condition had improved. The single exception was a markedly undersized girl with evidence of extensive bronchiectasis, who had progressively increasing dyspnoea, cough, sputum and haemoptysis. Of the 16 children examined none appeared invalid and most were in robust health. Râles were usually diminished in extent and few reported any sputum.—*Pulmonary Fibrosis in Children, A Report on 23 Cases, M. O. Raven, Lancet, January 9, 1937, ccxxxii, 80.*—(*A. P.*)

Hodgkin's Disease and Tuberculosis.—Eighty-three cases of lymphogranulomatosis were studied. Each case was tuberculin tested with 0.1 cc. of Old Tuberculin and avian tuberculin in dilutions of 1:5000; 1:2000; 1:1000; 1:100. In 87 per cent of the cases roentgenograms showed evidence of tuberculous changes, but these were either primary foci and obsolete, or single infiltrations of questionable activity. None of the cases of Hodgkin's disease complicated by tuberculosis showed any cavitation. Only 11 cases of the 83 had a positive tuberculin reaction and it was more marked to Old Tuberculin than to avian tuberculin.—*Lymphogranulomatosis and Tuberculosis, L. Funschtein, Probl. Tuberk., no. 4, 1936, p. 549.*—(*W. A. Z.*)

Hodgkin's Disease of the Lungs.—A case is reported which is of unusual interest because the clinical symptoms were limited to the lungs suggesting a pulmonary origin. The patient was a 42 year old man with X-ray evidence of a mass in the left upper lobe who successively complained of pain in the left shoulder, cough, loss of weight and fever. When first seen a diagnosis of carcinoma was made and X-ray therapy instituted. The first course of treatment was followed by marked clinical improvement and clearing of the area of increased density formerly seen in the roentgenogram. Pulmonary tuberculosis was then suspected but all sputum examinations were negative for tubercle bacilli. In the four years which followed, the patient received courses of X-ray therapy. Not until five years after the onset of his illness did he develop a superficial adenopathy suggestive of Hodgkin's disease. The case is illustrative of the confusion which may arise in the absence of lymph node, splenic or hepatic involvement which may lead to a diagnosis of lung tumor or pulmonary tuberculosis.—*An Unusual Case of Hodgkin's Disease of the Lung, S. Rubenfeld & E. Clark, Radiology, May, 1937, xxviii, 614.*—(*G. F. M.*)

Gaucher's Disease of Lungs.—This disease has been described as a nonhereditary

congenital, familial disease due to altered lipoid metabolism, with production of an abnormal substance called kerasin. Certain cells of the reticuloendothelial system are believed to store kerasin in what are known as typical Gaucher cells, chiefly in the spleen, liver, lymph nodes and bone marrow. Clinically the picture shows enlarged liver and spleen, conjunctival thickenings, a yellowish brown pigmentation of the skin and sclera, bone changes, leukopenia, thrombocytopenia, and a haemorrhagic tendency. There may be lymph node enlargement. The pain over the bones is a dull ache and there may be sharper pains over the spleen, liver and lungs. The most characteristic symptoms are the peculiar skin tint, splenomegaly and often hepatomegaly without ascites, flask-like expansion of the lower ends of the femora with dull ache or sharp pain, elevated temperature, leukopenia and hypochromic anaemia. The diagnosis may be clinched by splenic puncture and the finding of Gaucher cells or by finding them in excised glandular tissue. Sobotka and others have grouped three distinct types or clinical entities, with specifically differing chemical findings in certain tissues. Affection of the lungs seems to be rare and Myers finds no similar case described in British medical literature. *Case Report:* A girl of two and a half years was admitted to the Royal Waterloo Hospital, London, September, 1931, with enlarged abdomen, weakness and malaise. She was small for her age and the skin showed a slightly icteric tint. The spleen was greatly enlarged, reaching to the pubes, moderately firm and smooth, and the liver was enlarged two finger breadths. There had been severe epistaxes. The red blood cells were 800,000 per cmm. and the haemoglobin 19 per cent, the white blood cells 3,100. Fragility tests were normal and the van den Bergh test negative. The abdomen had been enlarged many months and many clinics had been visited. A splenic puncture was contemplated but decided against in view of the patient's desperate condition. Two days after admission Maingot, the surgeon, performed a splenectomy as a last resort. Typical Gaucher cells were found by the pathologists. The patient made an unexpected recovery and within a few months the blood count was normal. In April, 1932, she was readmitted with severe pain over the lower end of the right femur. Osteomyelitis was diagnosed at operation on the basis of finding a few colonies of staphylococcus and a little pus but this was not very convincing. During the next two years there was pain in the back and X-rays showed gradual atrophy of a number of vertebral bodies. Later there was inguinal and cervical lymph node enlargement of the lower end of the left femur. A flash like expansion persisted. In August, 1935, there was great substernal pain and severe cough, morphine being required. Signs suggesting lobar pneumonia appeared below the left clavicle. X-ray examinations showed a gradually spreading process from the mediastinum outward and fresh areas of bronchial breathing developed and persisted over many of which râles were heard. In November there was a right axillary pleurisy. There was extreme leucocytosis. During 1935 and 1936 the liver became enormously increased. Later, symptoms of nervous irritability appeared. Death occurred January 15, 1937, and necropsy showed characteristic findings. The Gaucher substance is nondoubly refractive and collections of cells containing it were present in all lymphoid tissue. It accounted for the early chest signs, the mediastinal and hilar nodes being grossly enlarged and the roots of both lungs extensively invaded. Also in many of the small subserous and intralobular lymph nodes the reticuloendothelial cells were loaded with Gaucher substance, producing small apparently solidified areas. There was no evidence of tubercle. A review of the literature did not disclose any similar case in England but one was published in France in 1933 by Merklen, Waitz and Warter in a patient 51 years old, without much emphasis on the pulmonary findings, except for Gaucher cells in the sputum, not found in the present case. Recently R. H. S. Thompson and G. P. Wright have supposed an "inborn error of metabolism" in which galactolipins play a rôle in the production and storage of kerasin.—*Gaucher's Disease of the Lungs, B. Myers, Brit. Med. Jour., July 3, 1937, 8.*—(*A. P.*)

Lung in Xanthomatosis.—Two cases of xanthomatosis with pulmonary changes are reported. The roentgenograms showed a miliary nodular infiltration, in the earliest stages, which gradually developed into fibrous patches. Histologically, as reported by other investigators, the nodules consist of lymphocytes and macrophages. Cells infiltrated with fat are found in the alveoli. Necrosis is not encountered. Other cases show marked gross and microscopical fibrosis and alveolar emphysema. The author believes that the lungs belong to the reticuloendothelial system. They filter out and bind lipoid substances from the blood, ridding the body of generalized xanthomatosis at the expense of progressive pulmonary fibrosis, emphysema and secondary embarrassment of the right side of the heart. The author further suggests that, though the aetiology of xanthomatosis differs so widely

from that of haematogenous tuberculosis, the roentgenograms are similar; the reticuloendothelial system is involved in both diseases and possibly the waxy capsule of the tubercle bacillus is a factor responsible for some of the similarities of the two diseases.—*On the Question of Involvement of the Lungs in Xanthomatosis, S. A. Reinberg & K. A. Moskacheva, Probl. Tuberk., no. 11, 1936, 1523.*—(*W. A. Z.*)

Periapical Empyema.—This condition is rare, only about 20 cases being reported since the time of Laennec, but since autopsy has been necessary for the correct diagnosis in several of these cases, many others must have been missed. The three following cases represent some of the diagnostic difficulties. Case 1 was a white man who at 66 years of age began to have progressive symptoms of cough, weakness, weight loss, night sweats, pain in the upper portion of the chest, multiple swellings at the base of the neck (6 months later), persistent drainage of thin pus (following incision) from sinuses bilaterally and in the sternal notch, anaemia, and episodes of fever during the fifth and last year of his illness. Expectoration was negligible and during four years in the sanatorium no tubercle bacilli were found; however, biopsy revealed tuberculous granulation tissue in the sinus walls. The leucocyte count was 15,000 with 77 per cent polymorphonuclears. X-ray revealed a clear cut, dense, half-spindle-shaped shadow over the upper lateral portion of the right lung field with the overlying intercostal spaces narrowed; slight haziness of the right upper and middle lung fields; right root markings moderately increased in size and density, and extending into the central lung field; the left lung clear except for slight increase in size and density of the hilum; and clear costophrenic angles on both sides. Two years later the only appreciable roentgenological change was that the homogeneous shadow extended to the third rib instead of the second. The only physical findings were dulness here, and pleural crepitations in the lower axilla. He died of congestive heart failure. Autopsy revealed a small brown heart and moderate amyloidosis, and reconstructed his pathological course. The primary lymphoglandular component (tracheal lymph node and a fibrous nodule in the right lower lobe, neither anatomically healed) progressed slowly and by local extension, or by lymphatic-haematogenous spread, involved the adjacent bony structures and the retroperitoneal lymph nodes. The tracheal lymph node was adherent to, and had ruptured into, the trachea leaving two small retracted scars. The pleura became involved from the osseous lesions, and finally the two pleural leaves fused into a mass of oedematous, hyalinized, fibrous tissue (2 cm. thick) walling off an apical pleural cavity, the viscous foul-smelling purulent contents of which escaped from time to time through the cervical sinuses. Case 2, a four months old girl, had coryza, cough and fever for a few days. After a short period of improvement there was a marked exacerbation plus weight loss, dyspnoea and cyanosis, and on hospitalization about seven weeks later there was dulness over the upper right lung field with patches of diminished breath-sounds anteriorly and tubular breath-sounds posteriorly and in the axilla. X-ray showed a slight shifting of the mediastinum to this side and a dense shadow apparently confined to the upper lobe. Because of the long course and the relatively low fever (101.8°F. rectally), tuberculosis was considered most likely in spite of the negative tuberculin test (0.01 mgm.) and 27,000 leucocytes with 80 per cent polymorphonuclears. A week later Cheyne-Stokes respiration developed; leucocytes were 56,800; the liver was 4 cm. below the costal margin; the whole right lung field was dull and breath-sounds were tubular high in the axilla, but there were no râles; and X-ray showed complete density of the upper lung field, a few hazy circular shadows throughout the lower lung field, and the costophrenic angle obscured. She died the next day and scattered patches of bronchopneumonia containing small abscesses were found in the upper and middle lobes. There was some atelectasis of the lower lobe. The empyema (300 cc. of thick pus containing *Staphylococcus aureus*) which was secondary to the pneumonia was confined to the upper and middle lobes and surrounded them like a thick mantle. Case 3, an eighteen months old boy, suddenly developed fever (102°F. rectally), mild pharyngitis, wheezing cough, and grunting respiration. The second day he had an obstructive type of breathing. The third day there was marked respiratory stridor with inspiratory retraction of the intercostal spaces; leucocytes were 11,000 with 58 per cent polymorphonuclears; scattered râles but no pulmonary consolidation were detected; bronchoscopic examination was normal; roentgenological examination revealed the mediastinum and heart to be normal except for a shift to the right, the right upper pulmonary field to be slightly hazy but the pulmonary markings visualized here, and the lower right and entire left lung fields (including the angles) to be clear. The fourth day stridor increased and he died. Both lungs were found to be diffusely congested and to contain, particularly in the dependent portions, minute areas of haemor-

rhagic bronchopneumonia. The exudate, which was mainly interstitial, consisted of mononuclear cells and erythrocytes. Pure cultures of Pfeiffer bacilli were found here and in the three ounces of thin sanguinopurulent fluid which surrounded the right upper lobe, being walled off by easily broken fibrinous adhesions. The inflammatory condition of the adjacent pleura may have caused some collateral oedema of the trachea. Periapical empyema is most often observed in young children and in them preëxisting upper lobe adhesions play a very small part. The early diagnosis is difficult chiefly because, in them, the clinical picture of pulmonary consolidation and of pleural effusion are much alike and may coexist. Periapical empyema may occur around the apex of a lower lobe. The effusion, limited to the area of a lobe, may cast a shadow of the same density as a lobar pneumonia and, also, radiopaque objects which are closest to the film cast the densest shadow. Roentgenoscopy and roentgenography with the patient in varying degrees of rotation and in different positions help to distinguish pneumonia and atelectasis from pleural effusion. When secondary symptoms prevent a clear cut diagnosis, exploratory thoracocentesis should be done without delay. The pathological factors which lead to periapical localization are not known but its rarity would be against any physiological cause such as the greater negative pressure in the upper portions of the pleural cavity. A variety of organisms have been responsible.—*Periapical Empyema, Report of Three Cases with Necropsy Findings, F. G. Kautz & M. Pinner, Amer. Jour. Roentgenol., April, 1937, xxxvii, 446.*—*(E. M. J.)*

Interlobar Shadows in Cardiac Failure.—During a period of decompensation the X-ray picture of the chest may show various changes. The hilar shadows tend to be accentuated. This is especially true of the right hilum in cases of mitral disease. Chronic congestion of the bases of both lungs may manifest itself as splotchy decreases in density of the lower lung fields. The diaphragm is clearly outlined, however, except in the presence of a pleural effusion. Pulmonary oedema in pure cardiac cases does not produce a striking X-ray picture, but in cases of cardiorenal disease with azotaemia there may be a fine mottling of the lung fields not unlike that seen in tuberculosis. In such instances a very pronounced pulmonary oedema is found at autopsy. Only a small proportion of infarcts are of sufficient magnitude to cast shadows on the X-ray film. Furthermore they are often obscured by a pleural effusion. The frequently made clinical observation of right pleural effusion has been verified by the X-ray. This may vary from a simple obliteration of the costophrenic angle to a massive opacity of the entire hemithorax. In more than one-third of cases of cardiac decompensation shadows of the interlobar fissures are seen on the X-ray film; they appear only after frank clinical signs of cardiac failure are present. Most frequently there is seen a fine transverse line passing from the hilar shadow horizontally across the lung field toward the axillary line. This shadow may be distinguished from those of simple trabeculae by the fact that the latter rarely approach the axillary line. Less commonly the shadow is several millimetres in thickness. Occasionally it is forked, and divides into a superior and inferior branch soon after leaving the hilum. Both branches meet the axillary line, but at different points. The shadow may be curved to form a sort of arch. Such bowing of the superior fissure is seen in tuberculosis. The author has observed this in cardiacs when there was a pleural effusion present. Rarely the shadow may appear double, one transverse line lying above and parallel to the other. The former corresponds to the superior fissure, and the latter which may be slightly curved corresponding to the oblique fissure. There are also instances in which, besides a horizontal shadow, there is a second shadow which runs diagonally downward and toward the midline coming off from the first. Very rarely such shadows are seen bilaterally. The fissure most commonly visualized is the lesser or transverse fissure, while the great or oblique fissure is least commonly seen. When the latter is visualized on the X-ray film the former is always seen. It is worth noting that the usual technique used in making X-ray pictures of the chest is most favorable for visualizing the transverse fissure. The exact location, size and shape of the fissures vary greatly in different individuals. The pathological changes present in these fissures vary from a simple subpleural oedema to interlobar adhesions. The latter may consist of only fibrin strands or may consist of fibrous bands which delimit accumulations of fluid. Usually, however, there is complete fusion of the two pleural surfaces. This may be a very feeble union or it may be of a thick fibrous nature. Islands of lymphocytes testifying to the essentially inflammatory nature of the process can be found here and there in this tissue. The horizontal fissure is rarely involved alone. Usually the two fissures on the right and that on the left are fused more or less completely even when only the horizontal fissure has been visible on the X-ray film. As for the greater pleura there is either an

effusion on the right side or on both sides, or a thickening of the visceral pleura of both lower lobes, or adhesions of varying degrees at one or both bases. The pulmonary parenchyma presents the well known picture. Tuberculous lesions are nearly always absent and if present at all are never active or recent. The effusions in cardiacs are habitually exudates, and contain always some fibrin. In 55 per cent of cases lymphocytes predominate in the fluid. In cases with pleurisy secondary to infarction polymorphonuclear leucocytes predominate. This group represents 20 per cent of this series. In 16 per cent endothelial cells predominate. Tuberculosis can always play a part in effusions in cardiac patients, but in fact it rarely does so. Inasmuch as the lung of the decompensated cardiac patient is never wholly free of an inflammatory process, one does not have to search far for a posssible source of the infection in the pleura. The impaired circulation in these patients undoubtedly reduces the resistance of the pleura to infection. The reaction of the pleura in each case is different, and may be either acute or chronic.—*Les images scissurales chez les cardiaques décompensés; leur fréquence et leur signification. Les "scissurites cardiques," C. Roubier, Arch. méd. chir. de l'app. resp., 1937, xii, 200.*—(*C. L. D.*)

Interlobar Effusion.—In the minds of most medical men, interlobar effusion is associated with pulmonary infections, especially pneumonia, lung abscess and tuberculosis. It may also occur as an aseptic process, cardiac failure being a common cause in such cases. This possibility should be kept in mind by those interested in diseases of the chest. Cases reported by Stewart, Kiser, Steele, Friedman and others are reviewed by the author and attention drawn to their conclusions. Steele felt that extensive adhesions were antecedent to the formation of interlobar effusions, Friedman emphasized that they should be differentiated from marginal pneumonia or malignancy, and Rigler concluded that they may arise as exudate from pleurisy or pericarditis in the course of active rheumatic fever following infarction or during cardiac decompensation. The diagnosis of interlobar effusion is facilitated by anterior-posterior and lateral views. The lateral view is especially important for in it a definite band or elliptical shadow is seen. No treatment other than treatment of the decompensation is required. Such effusions probably occur more frequently than is recognized. Three cases of interlobar effusion due to cardiac failure are reported.—*Interlobar Effusion Associated with Heart Failure, J. Levitin, Radiology, August, 1937, xxix, 190.*—(*G. F. M.*)

Diaphragmatic Hernia.—In 1912 only 15 of the 650 cases collected by Giffin had been diagnosed during life and by 1923 only 27 (Pancoast and Boles). The most recent case seen in the Truesdale Hospital, Fall River, Massachusetts, was examined by numerous physicians in several prominent medical centres and the diagnosis missed despite a clear cut history of adequate trauma and a flat chest film showing a large gas bubble above the left diaphragm. The fact should be recognized that this is not a rare condition and should be familiar to those dealing with disease of gastrointestinal and respiratory systems. On the roentgenologist rests the major share of responsibility for establishing the diagnosis and his opinion may be of invaluable assistance in directing treatment. The muscular portion of the diaphragm is divided into *pars sternalis*, arising from the posterior surface of the xyphoid process of the sternum, *pars costalis*, arising from the costal cartilages of the six lower ribs, and *pars lumbalis*, from the medical and lateral lumbocostal arches. The foramen of Bochdalek is situated posterolaterally where there is failure of fusion of the *pars costalis* and *pars lumbalis*, while the foramen of Morgagni is situated anteriorly where there is failure of fusion of *pars costalis* and *pars sternalis*. There is no sharp line of demarcation in the matter of fusion and even in the normal adult there is often a triangular space separating the muscle fibres of the posterior and lateral segments. This is filled by a thin layer of connective tissue which is less elastic and more easily torn than the adjacent muscle. That hernia is not more frequent is due to protection by the liver and perinephric fat. Thus, even in traumatic cases, 95 per cent occur on the left side. There are only 4 cases of true thoracic stomach in the literature, all having the findings originally described by Bailey. The literature, however, contains many reports of so called "thoracic stomach" that are really nothing more than the common type of hiatus hernia. True thoracic stomach is an anatomical variant which cannot be corrected surgically, in contrast to hiatus hernia. Hiatus hernia is by all odds the most common type of hernia through the diaphragm, usually occurring in overweight women over 40. These hernias are invested by a serous sac and represent displacements from a previously normal position. The oesophagus is of normal length but its point of entrance into the stomach may be high due to tortuosity. In most cases the hernia is not present in the erect position but is best

seen in the supine or Trendelenburg position with the right chest slightly forward. In some cases a gas bubble can be visualized above the diaphragm before giving any barium. Diaphragmatic hernia with short oesophagus has only recently been clearly recognized. The shortened oesaphagus militates against surgical repair. In these cases the oesophagus passes down through the posterior mediastinum almost straight and the upper end of the stomach, instead of being rounded, is more or less conical. The stomach is at least partly within the chest at all times and the displacement is visualized in the erect position. Careful observation of the first few swallows of barium will often differentiate this from hiatus hernia. In all the reported cases the junction of the oesophagus with the stomach was at the level of the seventh or eighth thoracic vertebra. Surgery is generally considered of little or no benefit. It is possible to bring the stomach down by stretching but it cannot be held. Congenital hernias show the greatest departure from the normal and the greatest variation. Their clinical course is often dramatic and death may ensue from asphyxia or intestinal obstruction. Because of displacement of the mediastinum and compression of the lung, symptoms may be predominantly respiratory. Most of them occur on the left through a persistent foramen of Bochdalek, less often through the parasternal foramen of Morgagni. The common type frequently includes large and small intestines, stomach, spleen, omentum, left lobe of liver, and sometimes other organs; if anteriorly, only bowel and omentum are involved. The diaphragm is frequently depressed and evidence of a sac is shown by a thin line of increased density. Traumatic hernias are not true hernias. Hedblom found about 90 per cent occurred in males and about 60 per cent from nonpenetrating injuries, of which 36 per cent were falls. About 95 per cent are on the left side and the tear runs radially from the central tendon, often to the periphery. The caecum is fixed in its normal position. Eventration of diaphragm may be congenital or acquired and results from aplasia or atrophy of muscle fibres. Excluding cases due to phrenic nerve operations it is rare, more common on the left side and in males. The diaphragm is elevated but intact and its mobility varies. Congenital absence of diaphragm is a very rare condition, with only 12 reported cases in the literature, several of these not definitely proved. It should be remembered that only mammals possess a diaphragm and it is not essential to life.—*Roentgen Ray Findings in Diaphragmatic Hernia, J. H. Marks, New England Jour. Med., October 22, 1936, ccxv, 777.*—(*A. P.*)

Traumatic Diaphragmatic Hernia.—A white male, aged 13 years, was admitted to the hospital two hours after an accidental gunshot wound of the upper abdomen and lower thorax. The bullet had entered the abdominal wall 2 cm. below the ensiform cartilage, passed in a transverse and superior direction, and emerged at the level of the eighth rib in the left midaxillary line. An X-ray revealed a comminuted fracture of the left ninth rib, fragments of the bullet in the soft tissues external to the fracture, but no gross changes in the pulmonary fields. The patient was not in shock, and made an uneventful recovery. He did however complain, during the first five days, of severe pain in the left shoulder. No significance was attached to this symptom, and a check X-ray of the chest upon discharge showed no change from the first plate. Eight months later he was readmitted in a moribund condition with a history of having, four days previously, fallen while skiing, with subsequent sharp pain in the epigastrium, referred to the left shoulder, followed by faecal vomiting three days later. Physical examination, laboratory studies, and X-ray suggested diaphragmatic hernia with obstruction, and this was confirmed by postmortem, the stomach and 18 inches of the midportion of the ileum being found in the left thoracic cavity. It is emphasized that it must be kept in mind that penetrating injuries of the lower thorax or abdomen may be associated with diaphragmatic injury, and that herniation and intestinal obstruction may occur immediately or later, due to various factors such as straining, exercise, childbirth, etc. In the presence of suggestive signs and symptoms of diaphragmatic injury, particularly shoulder pain, an exploratory operation might be more frequently employed to prevent disastrous sequelae.—*Traumatic Diaphragmatic Hernia, W. H. Mast & J. P. McDonough, Amer. Jour. Surg., November, 1937, new series, xxxviii, 371.*—(*A. E. M.*)

Mediastinal Hernia.—A discussion of the origin and mechanics of development of mediastinal hernia with cases illustrative of these is undertaken by the author because of the little material available on the subject in American or English literature. The frequent occurrence of the condition was not fully appreciated until the development of artificial pneumothorax and the attendant observations were made. Mediastinal hernia is the projection of one pleural space through the mediastinum into the other hemithorax.

It occurs most commonly in pneumothorax, either artificial or spontaneous, and is seen in one of two places, behind the sternum above and in front of the heart at the site of the atrophied thymus or below and posteriorly in the space bounded by the crura of the diaphragm, the oesophagus and the spine. The aetiological factor is a difference in pressure in the hemithoraces existing in the presence of a "weak spot" in the mediastinum. Thus, if the mediastinum is rigid there is no shift, if it is labile there is a shift *in toto* to the opposite side, but if there is a weak spot a hernia results. The hernia may be produced by either pulsion or traction. In the former type, which is also the most common, the hernia is increased in size by expiration and diminished in size on inspiration. This is due to the fact that the intrathoracic pressure on the side of the pneumothorax is higher on expiration, though possibly negative, than it is on inspiration and also higher than the pressure on the opposite side. The condition is usually initiated by cough or some similar incident producing a high positive pressure. Hernia due to traction is less common. Here the force producing the herniation is due to the loss of volume of one lung resulting in a great increase in the negative pressure in that hemithorax. Thus the normal lung is drawn through the weak spot. Fibrosis and atelectasis are chief among the factors producing this. The herniations due to traction are largest during inspiration. It is of real practical value to differentiate the two types. Occasionally a case may become reversed; thus it may change from a pulsion type of hernia as the result of a spontaneous pneumothorax to a traction type with the clearing of the spontaneous pneumothorax. In bilateral pneumothorax cases the presence or absence of hernia depends upon the pressure on the two sides of the mediastinum and may well give rise to the question as to whether there is too much air on one side or too little air on the other. A study of inspiratory and expiratory films will aid in answering the question by determining the type of herniation. Herniations in the lower "weak spot" occur in the presence of massive effusion and probably account for the triangular area of impaired resonance known as Grocco's sign. In all cases it would be well to study variations in the hernia as seen by films taken in inspiration and expiration.—*Mediastinal Hernia, R. T. Ellison, Radiology, November, 1937, xxix, 556.*—(*G. F. M.*)

Mediastinal Cysts.—The author presents a case of mediastinal cyst in which the cyst wall was lined with typical gastric epithelium and was perforated by a peptic ulcer. Four cases from the literature of mediastinal cysts containing gastric mucosa are reviewed. The author's case was that of a three and three-quarters year old male child who had been sickly and pale from birth. At three months of age he had passed tarry stools and was expectorating bloody mucous material. A short while previous to the time of the present study the child had begun to complain of pains in his chest associated with haemoptysis. On physical examination the child was seen to be extremely pale and emaciated. The blood haemoglobin was 40 per cent of normal. The thorax was broad and the respiratory excursion of the chest-wall was slight. Percussion disclosed a diminished resonance over the right lower chest. Near the hilum there were observed changes in resonance suggesting the presence of cavitation beneath. Coarse râles were heard over both lungs, especially in the region of the right hilum. Here also amphoric breathing was noted. Roentgenograms and tomographic studies were made. Two oval areas of decreased density partially involving the right half of the heart shadow were noted. One of these areas approximately 3.5 cm. in diameter was situated laterally to the other smaller area and was separated from it by what appeared to be a septum about one centimetre in thickness. There appeared to be an infiltrating tumor which had degenerated and broken down with the formation of cavities. An attempt was made to resect the tumor. The first-stage operation was uneventful, but during the second-stage operation severe haemoptysis developed. The patient died the same day. At autopsy it was determined that death was the result of haemorrhage from a penetrating peptic ulcer which had developed in a mediastinal cyst and had perforated into the lower lobe of the right lung. The cyst proper did not involve any of the lung parenchyma. It lay between the right lower lobe and the vertebral column. There was a chronic pneumonia in the right lower lobe. Elsewhere in the lung there was a well developed emphysema. The stomach which contained much blood was in other respects completely normal. There were two accessory spleens. Save for a slight scoliosis and some chronic swelling of the spleen no other abnormalities were present. The cyst wall resembled that of the fundus of the stomach. There was an inner circular layer of muscle and an outer more or less longitudinal one. Nerves and isolated ganglion cells were present. There was a loose subserosa with a normal appearing vascular supply. The submucosa was

scant and undiseased. The muscularis mucosa was of normal thickness. The mucosa itself was like that of a normal fundus with normal glandular structure and chief cells and other cell types in the usual distribution. Much of the superficial epithelium was missing. There was only one area with an increased number of round cells in the interstitium. One large defect in the wall of the cyst was present. Its edges were undermined and a certain amount of fibrinoid necrosis was present. This defect led into the lower lobe of the right lung. In the adjacent lung tissue evidence of chronic atelectasis, interstitial pneumonia and bronchitis was present. The cyst was entirely extrapleural. This case is the only one now on record where the lining of the cyst was composed entirely of gastric mucosa. In Staehelin's case there were various epithelial elements including ciliated epithelium, cylindrical cells, and cuboidal epithelium, as well as gastric epithelium. It was probably an oesophageal cyst which incidentally contained gastric mucosa. The cyst in the present case probably originated from a detached island of gastric mucosa or from an entodermal epithelial anlage of the foregut which had the potentiality of developing gastric mucosa. It is extremely unlikely that such a cyst is related to the tiny retention cysts developing from islands of gastric mucosa. One must bear in mind, however, the possibility of origin from displaced rudiments of the omphalomesenteric duct.—*Kongenitale mit Magenschleimhaut ausgekleidete Mediastinalcyste mit in die Lunge penetrierendem chronischem Ulcus pepticum, C. Böss, Virchow's Arch., August, 1937, ccc, 166.*—(*C. L. D.*)

Aschoff Bodies in Tuberculous Individuals.—In 1933 the authors reported finding in the heart of a twenty-eight year old female who died of pulmonary tuberculosis typical Aschoff bodies with multinucleated giant cells, and cells representative of all of the transitional stages between the myocyte and the Aschoff cell. There was no history of rheumatic fever nor was there any gross evidence of rheumatic endocarditis. Besides the typical Aschoff bodies there were observed interstitial and perivascular granulomata in which there was an irregular grouping of large pale basophilic cells with a very sparse infiltration of lymphocytes. There were other lesions which were more spindle or fan shaped. In these, the cells were arranged in parallel rows with connective tissue fibres between them. No acid-fast bacilli could be demonstrated in these nodules in spite of the fact that they were present in the tissue immediately beneath the endocardium, in the interstitium of the myocardium near blood and lymph vessels, and in areas of lymphocytic infiltration in the pericardium. In the tonsils there were granulomata not unlike rheumatic nodules, and others that more closely resembled the typical tubercle in structure. Similar findings in the hearts from two other cases of pulmonary tuberculosis led the authors to study the hearts from 215 cases of phthisis. A small block of tissue from the left ventricle was studied in each instance and where the findings were significant more extensive studies of the heart were made. In thirty-five of these hearts there were seen granulomatous formation in the connective tissue of the myocardium which differed from the usual tuberculous granuloma. These differences were consistent with hypothetical transitional stages between the tubercle and the Aschoff body. In eleven of these thirty-five hearts miliary tubercle formation could be demonstrated, whereas this was the case in twenty-eight of the remaining 180 hearts. In none of the thirty-five hearts was there any gross pathological evidence of rheumatic disease, nor was there any suggestion of a rheumatic history in the clinical records. Practically all of the cases in which these lesions could be demonstrated belonged in the age-group between ten and thirty. These granulomata were scarcely ever demonstrated in hearts from individuals dying of pulmonary tuberculosis before ten years of age or after forty. The majority of these hearts were from females. The rheumatic-like granulomata were seen in interstitial situations while the typical tubercles produced by haematogenous dissemination were localized in the region of the muscle fibres. The former lesions were perhaps produced by tubercle bacilli which had spread via the perivascular lymphatics. This could occur either by passage from the bloodstream via the coronary arteries, or from the endocardium, or even directly from caseated mediastinal nodes as was apparently the case in most of the preparations studied. It was apparent from this study that the distinction between the Aschoff body and the tubercle is only one of degree and extent, for all gradations between these two type lesions could be demonstrated. It therefore appears unlikely that there had been any specific rheumatic complication. The simultaneous occurrence of tubercles and rheumatic-like granulomata was observed in an old pericardial adhesion from a case of pulmonary tuberculosis with no clinical nor any anatomical evidence of rheumatic infection. In a study of nine hearts with clear cut tuberculous pericarditis there was found one such granuloma at the very centre of the tuber-

culous process. For comparison with the above the authors then studied in a similar manner 24 hearts from cases of rheumatic carditis. Sixteen of these were from females. Eighteen were from individuals dying between the ages of ten and forty. In the histories from eight of these cases there was a definite rheumatic story and in two of these eight there had been chorea. All of the hearts showed involvement of one or more valves. In twenty, Aschoff bodies could be demonstrated. These varied somewhat in their histological detail from case to case. In ten hearts they were classical. In five there were sparse accumulations of basophilic giant cells chiefly in the tissue spaces near blood vessels. This picture was a very common finding in the hearts from the tuberculous patients. In other hearts there were many hypertrophied myocytes, but there was a lack of basophilic staining and pleomorphism of the cells. Transitional forms were seen in various lesions. The similarity of many of the nodules to the interstitial granulomata seen in the hearts from cases of tuberculosis was very striking. In one of the hearts there were seen miliary tubercles. In this group of twenty-four cases there was macroscopical evidence of active tuberculosis in five. Healed tuberculous foci were seen in eleven, and in two of the remaining eight there was microscopical evidence of tuberculosis of the tonsils. It is evident that rheumatic disease can occur combined with various manifestations of tuberculous infection. In view of the fact that only a small percentage of tuberculous patients actually have rheumatic fever one is led to infer that the biological specificity of the reaction which forms the basis of rheumatic disease lies chiefly in some constitutional peculiarity of the individual rather than in a special kind of bacillus. Certainly the work of Löwenstein and Reitter offers no support to the latter hypothesis.—*Über das Vorkommen von Aschoffschen Knötchen in Phthisikerherzen, M. Masugi, S. Murasawa & S. Yä, Virchow's Arch., 1937, ccxcix, 426.*—(*C. L. D.*)

Cardiologic Departments in Tuberculosis Hospitals.—The author stresses the necessity of special cardiologic examinations for the tuberculous patient particularly in those cases where major surgical intervention is indicated. Besides the establishment of the surgical indication from pulmonary signs careful examination of the circulatory system plays an important rôle. The author feels that this side of preoperative care is rather neglected and therefore he suggests special cardiologic departments as permanent institutions in tuberculosis hospitals and sanatoria. Any surgical intervention requires an examination of cardiac efficiency, the determination of the vital capacity and of the respiratory coefficients, of the arterial and venous pressure, and of the circulatory velocity. Electrocardiographic, fluoroscopic examinations and metabolism tests should be carried out. Unlike any other surgical procedure a preoperative as well as postoperative regimen should be outlined in cooperative meetings between the pulmonary, surgical, and cardiologic service. The author's suggestion may well diminish the risks of thoracic surgery.—*Importancia de la Seccion de Cardiologia en los Hospitales de Tuberculosos, L. Ortega & Verdes, Rev. de Tuberc., Cuba, April, 1937, i, 257.*—(*F. G. K.*)

Haemothorax in Cirrhosis of Liver.—The accumulation of bloody fluid in the thorax is an extremely rare complication of cirrhosis of the liver. The patient observed was a man of sixty years of age with signs and symptoms of cirrhosis of the liver of alcoholic origin. In the latter part of his illness he developed a left-sided bloody pleural effusion which had to be repeatedly aspirated. Postmortem examination showed only cirrhosis of the liver without evidence of tuberculosis or neoplasm of the lungs.—*Bloody Pleural Fluid, an Unusual Complication of Cirrhosis of the Liver, H. A. Christian, Ann. Int. Med., May, 1937, x, 1621.*—(*A. A. E.*)

Oxygen Administration with Box Mask and Face Tent.—At present the common methods of oxygen administration are the closed tent (Hill, 1921, and Campbell, 1934) and the nasal catheter (Campbell, 1934, Marriott and Robson 1936). Both have definite disadvantages and are expensive, the running expense being about six to eight dollars a day. Campbell has introduced a box mask and small face tent which at least halve the expense if 100 cubic feet of oxygen a day are used. Also, no gas analysis is required and there are no valves to open and close, as with ordinary face masks. The box mask can readily be improvised from a cardboard box and a catheter. Aluminum masks are more durable. When in use the mask is connected with a Bobbin Flow-meter, oxygen passing from a standard automatic regulator attached to the oxygen supply cylinder. Moistening is unnecessary. It is possible with a flow of 6 litres per minute to maintain an oxygen pressure of 50 per cent of an atmosphere which ordinarily suffices but in cases of carbon monoxide poisoning almost pure oxygen is desirable for short periods to hasten dissocia-

tion of the carbon monoxide and haemoglobin. For this purpose a lint curtain is attached by an elastic band to the lower open end of the mask, hanging about four and one-half inches below the lower end. By this means, using a flow of 8 litres per minute the alveolar oxygen pressure may be raised to about 80 per cent. The *collapsible face tent* is a modified box mask but much lighter and thus valuable for aged or frail patients. Its wire framework is collapsible and covered with closely woven linen which can be moistened if the patient is hot or feverish. The efficiency of both mask and face tent depend on the end of the inlet tube (or catheter) being bent upward so that the incoming oxygen strikes the middle of the top of the apparatus.—*Oxygen Administration, Further Observations, J. A. Campbell, Lancet, January 9, 1937, ccxxxii, 82.*—(*A. P.*)

Periodic Accrediting of Households.—An economical method for detecting households that harbor patients with open tuberculosis was used in this study. The method included the use of the Mantoux test, but X-ray examinations of tuberculin-positive individuals were limited to those families where children were infected as well as adults. The Mantoux test using 0.1 mgm. of Old Tuberculin was applied to the members of 642 households in Minneapolis. The parents were tested in 57 per cent of the cases and the children in 84 per cent. There were 213 households where every member submitted to the test. Of 733 parents tested at an average age of 36 years, 57 per cent were sensitive to tuberculin. Of 860 children tested at an average age of 6.3 years, 5 per cent were sensitive to tuberculin. The annual attack rate of the parents was calculated to be 1.6 per cent, that of the children 0.8 per cent. On the basis of these calculations, the conclusion is reached that the average risk of infection faced by the children was distinctly lower than that faced in preceding years by the parents. In about 20 per cent of the 213 completely surveyed households, there were no tuberculin sensitive persons. In about 70 per cent, tuberculin sensitivity was limited to the adult members of the family. Since the children were tuberculin-negative, it was assumed that the open type of tuberculosis was unlikely in the infected adults. Consequently, these households were tentatively accredited as contagion-free units. Infected children were found in only 10 per cent of the 213 completely surveyed households. X-ray examinations, clinical and laboratory tests were concentrated on these 22 families. A source of tubercle bacilli was found in two of these homes.—*Periodic Accrediting of Households. An Economical Auxiliary Method for Controlling Human Tuberculosis, Suitable for Use in Private Practice, C. A. Stewart, Amer. Jour. Dis. Child., October, 1937, liv, 699.*—(*M. B.*)

Prevention of Tuberculosis among Young Adults in England.—Pulmonary tuberculosis is still a major medical problem, being responsible for the death of 25,000 persons annually in England and Wales. Among young persons of 15 to 24 its death toll is about two-thirds that for all other diseases together. In England the mortality figures show a satisfactory decline in all groups from the middle of the nineteenth century but latterly in young adults of 15 to 24, especially young women, the decline is less rapid and for 1930–1932 the mortality was only 4 per cent lower than in 1911, as compared with 36 per cent for women from 25 to 44. This marked decline began before any specific antituberculosis measures were taken while the recent check has occurred when interest in public health was at its height. This check began before 1914. Changing social conditions, operating especially among young adults, may be a factor. Housing, fatigue, nutrition and general hygiene are important factors. The importance of undernutrition agrees with the clinical observation that adequate diet is important in treatment and with the striking effects of food shortage in the late World War. The desirability of raising the level of national nutrition is now recognized. No amount of education can overcome a restricted food purchasing power of a substantial section of people. The League of Nations Committee on the Problem of Nutrition is now studying the question of availability of foodstuffs. As for housing, correlation between overcrowding and adult pulmonary tuberculosis has been demonstrated for various English county boroughs. The British Housing Act of 1935, following a special survey, sets up certain standards and encourages the reduction by local authorities of overcrowding. Rehousing, it is recognized, should not force the tenant to reduce expenditure for food, clothing and other necessities beyond the margin of safety. It may be stated that the average standard of living of English workers is still inadequate and especially so where conditions are substandard anyway. Many authoritative studies (for example, MacNalty, Burnet, Bentley) have demonstrated the importance of the economic factor in ultimate results. The major function of care and after-care organizations is to provide for the poor some of the more essential facilities enjoyed by the well to do. The baneful effects on the living standards of

whole families of the presence of a tuberculous member, especially the chief wage earner, are well known. To counteract these it may be necessary to create special insurance or assistance funds or to make grants in kind to these families, as in Sheffield, assuming rent payments during the period of disability and after adequate rehousing. Papworth and Preston Hall are examples of village settlements, but it has been estimated that not over 10 per cent of sanatorium ex-patients are suited to this type of life. Workshops and special sanatorium cottages exist, for example, in Wales and at Birmingham for the purpose of combining prolonged residence with instruction. However, the mass of patients will eventually have to return to a city environment and the chief hope is industrial coöperation. During the past decade or so, increasing attention has been paid to the risks run by adults, especially young adults, by prolonged contact with open tuberculosis, whereas formerly the emphasis was on the danger to children. Bentley showed that the incidence of new pulmonary cases in London for 1926–1935 was 7 per cent among adult contacts and 1 per cent among child contacts. In Philadelphia, Opie and others showed that the annual attack rate of manifest, that is, clinical, tuberculosis, in persons exposed to sputum-positive cases, showed its greatest increase four to five years after the commencement of known exposure and the maximum excess mortality about two years later. Lloyd and MacPherson at the Brompton Hospital in London (1936) studied the case records of a series of clinically tuberculous young adults, aged 15 to 24, to determine the time relationships with the termination of known exposure to an earlier case, evidence of which was found in 40 per cent, 82 per cent of those in the home. Clinically recognizable tuberculosis during adolescence or adult life plays an important part in producing tuberculosis in young adults. Medical students and nurses are among those apparently subject to increased risks. Heimbeck, at the Ullevaal Municipal Hospital at Oslo, found an unusually high incidence in previously tuberculin-negative probationers of pleurisy, erythema nodosum and even meningitis, and Mariette and others in the United States have found troublesome accompaniments to first infection among nurses. Known exposure to contact in adult life should be reduced to a minimum. Those unavoidably in contact with open tuberculosis should be kept under observation, especially young adults and especially those whose tuberculin reactions have recently become positive. Even after termination of such exposure observation should be continued several years. More expeditious diagnosis and removal of sputum-positive cases for segregation and treatment is most important to reduce contact infection. In the cases of medical students and nurses hygienic precautions should be taken but more promising is BCG vaccination. Periodic examination during and after exposure is most important. The ratios of new cases to deaths give a rough indication of the change in average prognosis over the past two decades. For England these ratios have decreased but little between 1913 and 1922, and between 1922 and 1935, hence one can scarcely assert that the average prognosis has improved materially, the declining mortality being due mainly to the declining incidence of new cases. The prognosis of the average case has not materially changed for thirty years although that of a small selected group treated by collapse therapy has considerably improved. Three methods of improving prognosis are by earlier diagnosis, by better treatment, and by better after-care. Recent surveys also show that the proportion of cases diagnosed in the slighter or earlier stages has not materially increased over twenty or thirty years. The proportion found suitable for pneumothorax treatment in England is relatively low, 10 per cent in the London County Council series. Their chances of survival at the end of five years were found improved by 20 per cent, the best results obtaining on those with strictly unilateral lesions, only one-quarter of the total group. Thus, the effect of pneumothorax therapy on average prognosis is not over 4 per cent, and can be increased only if the proportion of suitable cases can be increased. Methods for improving early diagnosis have heretofore been directed toward shortening the interval between the onset of symptoms and the recognition of progressive lesions. But before definite symptoms and signs appear progressive lesions are recognizable only roentgenographically. What is required, therefore, is a procedure for discovering these lesions in the preclinical stage. This involves periodic X-ray examinations of the supposedly healthy. For economy of effort, such groups as students and factory workers are most suitable for such investigation. Periodic examination of contacts requires first the listing of known cases, which may come to light often too late to prevent spread of infection. However, the proportion of home contacts examined is increasing. Group schemes of periodic examination have been tried in the United States, usually among young adults, with a higher than average case incidence. They combine roentgenography with tuberculin testing. Periodic ex-

amination of employees has been developed especially by Fellows and others at the Metropolitan Life Insurance Company. In London, England, at the University College Hospital, routine examination of students has been inaugurated, serial roentgenography being the basis of the method. The rationale of this method rests on the hypothesis that, on the average, a slight and early case does better with than without treatment and responds better than a later and more extensive one. The occasional risk of unnecessary treatment must be assumed provided efforts are made to obviate economic hardship. To reduce expense one may utilize such methods as the Powers X-Ray Tuberculosis Travelling Survey Service, a "triumph of American mass production," which takes paper films at the rate of 150 an hour and at cost of about seventy-five cents a piece. Beginning with such easily accessible groups as students and nurses, one might proceed to young State employees and industrial groups; where a low infection incidence prevails, a preliminary Mantoux test should be included.—*Prevention of Pulmonary Tuberculosis among Adults in England in the Past, Present and Future, P. M. D'A. Hart, Lancet, April 24, May 1, and May 8, 1937, ccxxxii, 969, 1033, 1093.*—(*A. P.*)

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